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# FILARIASIS WITH TESTICULAR INVOLVEMENT

## REPORT OF A CASE

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In view of the large amount of migratory tropical native labour employed on the gold mines it was anticipated that a fair number of cases of filariasis would have been reported. Search of the available literature did not lend support to this assumption and it was accordingly felt that the following case might prove of interest.

The patient, a tropical native aged about 34 years, was admitted to hospital on 1 August 1949 with the complaint of testicular pains of three days' duration. He stated that the pain was of a nagging nature and worse in the erect posture; also that the testis was getting larger. There was no history of injury and venereal disease was denied. Examination of his mine weight card showed that he had lost 9 lb. in weight since his engagement six months before admission. He had never had any similar condition nor had he noted a like condition among acquaintances at his kraal. His home was at Mwaya location in Tanganyika, and he passed Lake Nyassa to and from his home.

*Examination*: *General*. Heart and lungs appeared normal. Abdomen negative, no splenomegally. Blood pressure, 120/80 mm. Hg; he was apyrexial.

*Scrotum*. The scrotal skin appeared normal but for the most dependent and posterior part on the right side where it was adherent to the epididymis and showed induration and oedema. Apart from this site, it moved freely over the underlying structures. The hernial orifices were closed and no impulse was transmitted. The contents of the scrotum on the left side appeared normal. The right testis was tender and apparently twice the size of the left, and there were areas of softening in its substance. The epididymis was craggy and markedly enlarged, the maximal enlargement being in the region of the globus major. The sinus epididymis was partly obliterated. There was no rotation of the testis, the epididymis lying posteriorly. The cord was much thicker than on the left, and a number of nodules the size of a match head were palpable near the epididymis.

*Rectal examination* showed that the prostate was small and firm and painless, and the vesicles were impalpable.

The *blood count* on 6 August 1949 revealed a slight normochromic anaemia but no eosinophilia. Haemoglobin, 12.3 G.%. Colour Index, 0.94. Erythrocytes, 4.1 million per c.mm. Leucocytes were 5.400 per c.mm. and of these, neutrophils were 57%, monocytes 9%, lymphocytes 33% and eosinophils 1% (South African Institute for Medical Research report 439625-49).

X-ray of the chest showed no evidence of tuberculosis or other pathology.

He was kept in hospital under observation for 14 days and during this period he was apyrexial but the testicle appeared to be enlarging. The areas of fluctuation in the body of the testis were more marked and the pain had become worse and interfered with his sleep. The area of induration in the scrotal wall was more pronounced, and an area of fluctuation could be felt below it.

A diagnosis of tuberculous epididymitis with extension to the testis was made and operative treatment decided upon.

*Operation*. Under local infiltration and block anaesthesia the testis was delivered through a 5-inch inguinoscrotal incision. The cord was divided and the testis removed. A small rubber drain was inserted and the remainder of the incision closed with interrupted sutures.

*Gross Pathology of Operation Specimen*. The epididymis was enlarged, firm and nodular, but there was no evidence of caseation or abscess formation. The testis was enlarged but otherwise appeared to be normal.

*Histological Examination*. This revealed the appearances of filariasis (Figs. 1 and 2) (South African Institute for Medical Research No. 442124/49).

*Subsequent Investigations*. A specimen of blood taken at midnight revealed the presence of *Microfilariae bancrofti* (S.A.I.M.R. 344011/49). Subsequently I was able to demonstrate living microfilariae in a similar specimen. Unstained serum from the clotted blood was

examined under a coverslip using the low power objective. The parasite is large and occupies almost the length of the low power field. Unstained it is structureless and resembles a worm.

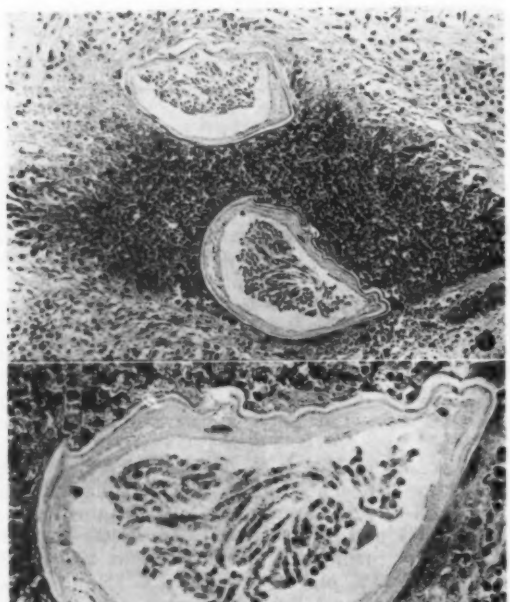

*Fig. 1.* This shows the presence of testicular tissue with transverse sections of two filariae. These are surrounded by abscess formation, and within them can be seen the microfilariae
*Fig. 2.* This is an enlargement of part of Fig. 1 to demonstrate more detail.

*Subsequent Progress.* The drain was removed on the third post-operative day and the wound healed by first intention. He was returned to his normal work. Two months after operation there was no oedema of the scrotal wall and no residual mass or other abnormality could be felt in the scrotum. The contents of the left side still remained apparently normal.

## DISCUSSION

Filariasis is a condition caused by infestation with the *Wüchereria bancrofti* or *malayi*. It is more prevalent in males, and is endemic in Japan, China, the Philippines and the United States of America. In the African continent it is very common in the northern part of Lake Nyassa (where this case is presumed to have contracted the disease) but less prevalent in the southern part. There are numerous vectors, but the common one for *W. bancrofti* is *Culex fatigans* and for *W. malayi* is a genus known as *Mansonioides*.

The adult worms inhabit the lymphatics. They are threadlike and the female is about 3 inch to 5 inch long and the male half that length. The female is ovoviviparous and is almost entirely twin oviduct terminating in a vagina. The *Microfilariae sanguinis hominis* are living larvae inside the elastic shell and are present in the blood stream in large numbers about eight months after infestation. They are mainly discovered at night in the peripheral blood as the condition is spread by night-biting mosquitoes. By day the microfilariae inhabit the pulmonary capillaries. In the mosquito's stomach the ingested blood coagulates and the larvae migrate through the wall of the gut and make their way to the region of the proboscis. The cycle in the mosquito takes fifteen days and a large percentage of the mosquitoes die, especially if the infestation is heavy. When the victim is bitten the larvae make their way into the skin and enter either via the puncture or the hair follicles. In one bite 30 to 40 parasites may be acquired but, unless the infestation is heavy, no symptoms are engendered. This is probably why there have been no cases reported in the Transvaal, although we have the vectors and a reservoir of microfilariae in our migrant native population.

De Meillon (1942), reporting on blood smears taken at random from patients at the Witwatersrand Native Labour Association's Hospital, found that of 102 blood slides examined four showed the *W. bancrofti*. Of these two cases came from Southern Rhodesia and two from Portuguese East Africa. This investigation suggests that there is a number of tropical natives on our gold mines infested with microfilariae who exhibit no clinical manifestations.

Cawston (1935) reported a case where chyluria in pregnancy resulted in the discovery of microfilariae. There were no other symptoms and he considered that the condition had arisen in Natal, despite the fact that this patient had had previous residence in India. According to him microfilariae have not been discovered in the mosquitoes of South Africa, although there are numerous strains of possible vectors. In Durban only six out of more than 30,000 blood smears revealed the presence of filariae or spirillae. Most of these smears had been taken by day and Cawston considered that if more had been taken at night the incidence of microfilariae would have been appreciably higher.

Man is the only vertebrate host for *W. bancrofti* but *W. malayi* have been found in some monkeys.

*Symptomatology.* Unless the infestation is heavy there are usually no symptoms. The disease manifests itself according to one of the following well-defined groups which may, however, overlap:

1. *Filarial lymphangitis.* The worms obstruct the lymphatics and predispose by stasis to secondary infection. The infecting organism is usually the *Streptococcus haemolyticus*, and the symptoms and signs are those of a septic lymphangitis. This may resolve and leave no trace or develop into a septicaemia. The lymphatics may remain thickened and the filariae may occasionally be detected therein. The lymphatic glands are usually enlarged and abscess formation may occur in the deeper structures (e.g. the kidneys or muscles).

2. *Lymphoma.* The lymphatic glands, especially of the groin and inguinal region, are enlarged and elastic, tending to diminish in size at night. Orchitis may occur but this is usually of short duration. Of 14 cases reported by Howard (cit. Smith), seven resolved but seven suppurated. Other authorities, however, consider suppuration to be very rare. A lymphomatous varicocele may occur due to obstruction of the lymphatics of the spermatic cord. Both these sequelae are usually associated with hydrocele. A pedunculated cystic swelling may occur on the cord—a pedunculated lymphoma—which resembles an epiploic hernia and is reducible.

3. *Chylous effusions.* These are due to obstruction of the thoracic duct and back pressure on the lymphatic vessels. Chylous ascites and haematochylurea may occur. The urine clots on standing and is milky in appearance. Lymphscrotum or lymphvulvae may occur, and due to the tension may result in exudation through the skin and secondary infection.

4. *Elephantiasis.* Elephantiasis is due to the blockage of the lymphatics. A collection of oedematous fluid in the tissues stimulates the fibrous subcutaneous tissue to proliferate, the skin becomes coarse and discoloured, and the superficial veins distend. Elephantiasis of the scrotum is common but the penis is rarely involved. The lower limbs, breast and upper limbs may be involved.

The life span of the adult filaria is considered to be about nine years, and to date there is no treatment of proven value for eradicating it. The microfilariae can however be eradicated from the blood stream by a number of drugs. Among these drugs may be mentioned hetrazan, fouadin and anthiomaline. Recent work indicates that hetrazan may have some lethal effect on the adult filariae. The eradication of microfilariae, which in themselves cause no symptoms, whilst the adult female remains as a source of supply, seems pointless.

There are numerous procedures for elephantiasis but fewer for the chylous effusions. Bladder obstruction in chyluria may need lavage and any associated anaemia need appropriate therapy. Chemotherapy is of value in dealing with, and in the prevention of, secondary infection.

In this case of filariasis there was no eosinophilia nor any clinical indication of the aetiology.

I wish to thank Dr. J. Murray and his staff at the South African Institute for Medical Research for preparing the excellent photomicrographs and allowing me to make use of them.

I am indebted to Dr. A. J. Orenstein, Chief Medical Officer, Rand Mines, Limited, and Dr. C. Berman, Senior Medical Officer, Consolidated Main Reef Native Hospital, for permission to submit this paper for publication, and to the latter also for constructive criticism and advice.

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## ABSTRACTS

Nagley, N. M., and Logg, M. H. (1949): *Para-aminosalicylic Acid in Tuberculosis*, Lancet, **256**, 895.

Nagley and Logg analyzed 37 cases of active pulmonary tuberculosis treated with para-aminosalicylic acid during 1947 and 1948. Observations in these and other cases indicate that para-aminosalicylic acid is especially valuable in the acute exudative type of disease, particularly in toxic febrile patients. Such effects as reduction in temperature, fall in erythrocyte sedimentation rate, gain in weight and a feeling of well-being occur long before radiologic improvement can be demonstrated, and, once present, they remain during treatment. Treatment with this drug often makes the patients fit for collapse therapy. The tuberculostatic action of para-aminosalicylic acid cannot be doubted. The mode of action is still uncertain. Youmans inclines to the view that the drug alters pulmonary tissue reaction from exudative to proliferative by an effect on the enzymic mechanism of the tubercle bacillus. The authors are impressed with the direct action which para-aminosalicylic acid seems to exert on the host, as shown by the patient's feeling of well-being, the antipyretic effect and the improvement in the general condition. The morphologic alteration of the bacilli which is definitely seen under the microscope, points to a direct effect also on the bacilli, while the radiologic changes in the lungs support Youman's view. The low toxicity of para-aminosalicylic acid and the fact that it does not seem to produce resistant strains give it advantages over streptomycin. However, patients with acute postoperative and other types of acute pulmonary tuberculosis should not be denied streptomycin, but the value of para-aminosalicylic acid in such cases, either in conjunction with streptomycin or after its use, should be realized. Para-aminosalicylic acid does not necessarily produce a 'cure', but it often may prepare the patient for collapse therapy of pulmonary surgical treatment.

Wilson, W. Weatherston (1949): *Injection Treatment of Hydrocele.* Lancet, **256**, 1048.

According to Wilson the treatment of hydrocele by aspiration and injection in the outpatient clinic is preferable and superior to treatment by operation. He describes his technique as follows: The scrotum is washed and shaved, and the patient lies supine upon a couch. Transillumination is used to confirm that the testis is lying posteriorly. To a small area of scrotum over the upper pole of the hydrocele tincture of iodine is applied; this is not painful, provided that the scrotum is not swamped with iodine. A weal of 2% procaine is next raised, and through it a large aspirating needle is thrust into the tunica vaginalis, care being taken to pass it between visible blood-vessels. Fluid is expressed by squeezing gently on the scrotum, as much as possible being evacuated, for if any is left behind it dilutes the sclerosant and renders it less effective. At this stage the testis and epididymis are carefully palpated to exclude an underlying pathological lesion. Whether the aspirated fluid is that of a hydrocele or of a spermatocele, 6 ml. of quinine urethane is injected and is massaged round the sac. The needle is withdrawn, and the puncture is sealed with collodion and a wisp of cotton-wool.

The injection is usually not felt by the patient, but slight transient pain referred to the tesis, groin or loin may occasionally be felt. The patient is sent home with instructions to report in a month or to return earlier should pain occur.

A second or third injection may on occasion be required, and in a loculated hydrocele each loculus requires a separate injection. Final assessment of the result should not be made until six months have elapsed, for fluid may persist up to that time and undergo resolution of its own accord.

Quinine urethane is not toxic in the tunica vaginalis, since it is not rapidly absorbed from there.

In 16 out of 18 patients thus treated one injection sufficed, in one two injections were given and in another three injections. Only in this last case did fluid re-accumulate in spite of three injections; the patient has since been radically cured by operation.

Guggisberg, H. (1948): *Die medikamentöse Leitung der Geburt.* Cahiers mensuels de Médecine, Schwarzenberg (Switzerland), **4**, 477.

Small doses of quinine heighten the tonus of the uterus in labour, thus promising the best results in hypotonically inhibited contractions. The uterus, sensitized by quinine, easily reacts upon different stimulants, also upon small quantities of hypophysin. Therefore, quinine is very useful in the induction of labour, when combined with other drugs, especially hypophysin.

It is an important fact that oral administration of small doses of quinine gives the best results; there is no danger of unpleasant secondary effects.

In the Cantonal Hospital for Women at Bern (Switzerland) the following method is practised in uterine inertia: 0.1 gm. of quinine sulphate, followed after an hour by ¼ VU (= Voegtlin unit) of hypophysin, which dose may be eventually increased to 2 VU at the utmost.

For induction of labour: 10 mg. of ovocyclin (= oestradiol); the next morning (e.g. at 6.30 a.m.) 30 gm. of 01. ricini; at 7 a.m. an enema; 8 a.m. 0.2 gm. of quinine sulphate, repeated at 8.30; at 9 a.m. a hot bath and 1 VU of hypophysin; 9.30 a.m. 1 VU of hypophysin, repeated at 10 a.m.

Van Deinse, J. B. (1949): *Drug Treatment of Ménière's Syndrome.* Nederl. Tydschr. v. Geneeskunde, **93**, 2619.

I. *Drugs acting on the vascular system.* Histamine is recommended nowadays, especially by American authors. The Otological Department of the Amsterdam University prescribes this drug in the following way: On admission to hospital the patient is given an intravenous infusion of 1.7 mg. histamine in 200 $cm.^3$ of isotonic salt solution, every second day. This is repeated 6 times. Ambulatory after-treatment consists of subcutaneous injection of 0.1-0.3 mg. histamine, 2 or 3 times a week. If the patient is free of symptoms after 2 or 3 months, the injections are administered with greater intervals.

II. *Drugs which increase the excitability of the labyrinth.* Charcot was the first to administer quinine, in doses of 1 gm. daily. The Amsterdam Otological Department uses pills, containing 125 mg. quinine base and 15 mg. phenobarbital (luminal), two pills being given every night.

One may also give a subcutaneous injection of 5 mg. pilocarpine hydrochloride. If this is well tolerated, daily injections of 10 mg. are administered for 14 days.

Another possibility is: injections of 0.5 mg. of strychnine sulfate.

III. *Drugs which decrease the excitability of the labyrinth.* These are: atropine (twice daily 0.25 mg.), bulbocapnine hydrochloride (100 mg. daily in tablets); or methenamine (urotropin) 500 mg. daily, with 100 mg. barbital (veronal).

The latest drug to abolish the function of the N. octavus is streptomycin. The Amsterdam clinic treated a few patients with 2 gm. daily. After 3 or 4 weeks the labyrinths have lost their excitability, and treatment is stopped.

Blackie, W. K. (1949): *Quinine in Acute Malaria.* Lancet, **257**, 261.

The author, practising in Southern Rhodesia (Africa), writes: 'The prevailing tendency amongst writers on the treatment of malaria is to stress the virtues of the newer plasmocidal drugs, while showing remarkable indifference to the established properties of quinine. This attitude is also prevalent amongst newcomers to medical practice in the tropics—so much so that one is obliged to conclude that the present-day teaching in tropical medicine fails to stress the salient fact that quinine is still our sheet anchor in the treatment of acute malaria.

Those of us who through years of practice in the tropics have learned to respect the unpredictable vagaries and viciousness of malignant malaria in the non-immune, continue to regard quinine as the most reliable and most effective of all the plasmocidal drugs in gaining speedy control of an acute attack; and this still holds good after giving fair trial to all the newer plasmocidal drugs. Furthermore, the prejudice expressed in some authoritative quarters against the use of parenteral quinine therapy—more especially against intramuscular injection—is wholly unjustified. With the patient prostrated by the persistent and intractable vomiting and the profound toxaemia that so often accompany attacks of acute malignant malaria, oral therapy is wholly unreasonble; in such circumstances the parenteral administration of quinine will not only save life but will spare the patient hours—even days—of distress and suffering.

I consider that the basic procedure in the treatment of acute malaria in Africa consists in: (1) overwhelming the infection and so gaining rapid control of the attack with quinine either orally or, in special circumstances, parenterally (quinine acts promptly on the parasites in the early stages of schizogony), and (2) following through with a full course of one of the newer plasmocidal drugs. Dosage varies with the strain or species of the causal plasmodium.'

These words, written by a man of experience, certainly deserve wide attention. One should like to ask, however: why switch over to 'one of the newer plasmocidal drugs' when the infection has been controlled? Why not stick to the 'sheet anchor in the treatment of acute malaria', as Blackie expressed it himself?

Zarafonetis, C. J. D. (1949): *Therapeutic Possibilities of Para-amino-benzoic Acid.* Annal. Int. Medicine, **30**, 1188.

The first important clinical use of PABA followed demonstration of its value in the treatment of several of the rickettsial diseases. More recent investigation has shown encouraging results in a number of diverse conditions of unknown etiology, and indicates that further investigation is justified. These include: lymphoblastoma cutis, certain forms of lupus erythematosus, active dermatomyositis, scleroderma and dermatitis herpetiformis.

PABA was also seen to be capable of producing a striking fall in the leukocyte counts in patients with chronic myelogenous leukaemia. The drug is best tolerated as a neutral salt, and in the Department of Internal Medicine, University of Michigan, it was given as a 10% solution of the sodium salt (NaPAB), in doses of 10 to 40 $cm.^3$ (1 to 4 gm.) at intervals of 2 to 3 hours in milk, fruit juice or ginger ale. PABA is rapidly excreted in the urine; the optimal dosages have not yet been determined.

In leukaemia the lowering of the leukocyte count could be maintained only through continued administration of large quantities of PABA, while clinical improvement was slight and temporary. The author does not consider therefore that it is of practical value in leukaemia. In lymphoblastoma cutis 6 patients treated experienced relief from pruritus and objective improvement of the skin. Treatment was discontinued because of the development of oedema with the sodium salt, but in two cases where the potassium salt was given this did not occur. Thirty-three cases of lupus erythematosus were treated. The best results appeared to have been obtained in the chronic and subacute disseminated types. It was noted that the incidence of unfavourable reactions was higher in lupus erythematosus than on other diseases.

In dermatomyositis 4 cases out of 5 treated showed improvement. One case which responded dramatically was alive and active after being maintained for 18 months on KPAB.

Five patients suffering from scleroderma were treated with good results. The author's observations led him to the view that the wall of the intestinal tract softened under treatment, as the skin did, which might result in intestinal dilatation. He advises therefore that all patients with scleroderma submitted to PABA therapy should have a preliminary complete roentgenization of the entire gastro-intestinal tract and the treatment should begin cautiously with 4 to 6 gm. per day.

In highly resistant cases of dermatitis herpetiformis PABA was of much value as long as its use was continued. The author indulges in no speculations as to the possible mechanism involved in the effects of PABA in lymphoblastoma cutis, scleroderma and certain cases of dermatomyositis. Toxic reactions were: drug fever and dermatitis medicamentosa, and there was one of toxic hepatitis. Nausea is the most frequent reaction, and a reducing substance, probably not glucose, occurs in the urine of all patients taking large doses of PABA.

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## EDITORIAL

### SPECIFIC ANTIGENS OF CHOLERA VIBRIO

#### COMPLETE ANALYSIS AND PRACTICAL APPLICATIONS

Dr. J. Gallut of the Institut Pasteur, Paris, has made an exhaustive analytical study of the heat-stable antigen of the cholera vibrio (specific O antigen), and his findings have recently been published in the Bulletin of the World Health Organization.[1] Confirming research work carried out by Burrows, the author notes that a greater number of O antigenic factors exists in cholera vibrios than has so far been admitted. As many as 13 antigenic factors (A, B, C, . . . M) can be detected. This discovery is very important, as it may enable the accuracy of the serological diagnosis of cholera to be increased, and vaccines to be prepared in such a way as to be better adapted to the various epidemics.

The author analyzed 82 strains, which fall into two groups; the first comprising 61 so-called 'agglutinable' vibrios, and the second comprising 21 so-called 'inagglutinable' vibrios. Following the usual serological diagnosis, the 61 'agglutinable' vibrios were classified as follows: 2 Ogawa, 40 Inaba, 18 Hikohima and 1 atypical. Complete antigenic analysis, however, revealed their multiple O factor content and showed the antigenic differences which may appear between vibrios considered as serologically identical. Antigenic analysis of the second group revealed the presence, in some of the vibrios, of factors found in the first group. Moreover, there are frequency variations in the distribution of these factors, which indicate an essential point: factor A, which is to be found in all 'agglutinable'

1. Bull. World Hlth Org., 1949, **2**, 39

## VAN DIE REDAKSIE

### SPESIFIEKE ANTIGENE VAN CHOLERA-VIBRIO

#### VOLLEDIGE ONTLEDING EN PRAKTIESE TOEPASSING

Dr. J. Gallut van die Institut Pasteur, Parys, het 'n grondige ontledingstudie van die teen hitte bestande antigeen van die cholera-vibrio (spesifieke O-antigeen) gemaak en sy bevindings is onlangs in die *Bulletin of the World Health Organization*[1] gepubliseer. Hy bevestig navorsing wat deur Burrows gedoen is en wys daarop dat 'n groter aantal O-antigeenfaktore in cholera-vibrios voorkom as wat tot dusver erken is. So veel as 13 antigeenfaktore (A, B, C, . . . M.) kan bespeur word. Hierdie ontdekking is baie belangrik aangesien dit groter noukeurigheid met serologiese diagnoses van cholera moontlik kan maak asook die bereiding van entstowwe op 'n wyse wat beter sal aanpas by die verskillende epidemies.

Die skrywer het 82 soorte ontleed wat onder twee groepe ressorteer: die eerste behels 61 sogenaamde bindbare vibrios en die tweede 21 sogenaamde onbindbare vibrios. Volgens die gewone serologiese diagnose is die 61 bindbare vibrios soos volg ingedeel: 2 Ogawa, 40 Inaba, 18 Hikohima en 1 afwykend. Volledige antigeniese ontleding het egter hulle veelvoudige O-faktor-inhoud aan die lig gebring en die antigeniese verskille getoon wat tussen vibrios kan bestaan wat as serologies identiek beskou word. Uit antigeniese ontleding van die tweede groep het geblyk dat in sommige vibrios faktore aanwesig is wat in die eerste groep gevind is. Bowedien is daar herhalingsverskille in die verspreiding van hierdie faktore wat 'n allerbelangrike feit aandui: faktor A, wat in alle bindbare vibrios aangetref word, is nog nooit in onbindbare vibrios gevind

1. Bull. World Hlth Org., 1949, **2**, 39.

vibrios, has never been found in 'inagglutinable' vibrios. For this reason, the author is in a position to state, in agreement with Burrows, that antigen A is the only specific antigen of the authentic cholera vibrio (and of the El Toro vibrio, which is serologically identical). Experience has also shown that factor A can exist alone, thus making it possible to define a new and fourth type off cholera vibrio. Factors B and C are essential, as they are specific for the Ogawa (B) and Inaba (C) types, but only in a subsidiary way, because of their relationship with factor A. Factors B and C, were, in fact, encountered more frequently in non-cholera vibrios. Factors D, E, . . .M are of less significance. The O antigenic formulae of the 'agglutinable' vibrios, and the frequency of O antigenic factors in both groups of vibrios, are summarized in two tables.

These experiments may have important practical consequences: (*a*) In diagnosis, anti-O monospecific serum A will have to be used in future in preference to Ogawa and Inaba sera, which are prepared from strains of which the antigenic formula is only partially known. Agglutination due to non-specific factors will thus be avoided. (*b*) Complete antigenic analysis of the cholera vibrio may change present views on the preparation of vaccines. In this respect, too, the accepted conception of the Ogawa, Inaba and Hikojima types now seems outmoded. If the need arises of using a so-called 'monovalent' vaccine to combat an epidemic of a supposedly 'single' serological type, it would be advisable to proceed with the greatest possible care and not rely too much on the respective mono-specificity of the three classical types.

Antigenic analysis showed that Egyptian cholera vibrios of the 1947 epidemic, nearly all of the Inaba type, contained all the factors except B, G and J, with factors A, C, D, E and L predominating. For this reason, Dr. Gallut is of the opinion that not only does a mixed vaccine composed of one Ogawa and one Inaba strain appear to be inadequate, but a vaccine composed of the Inaba type alone might be partially ineffective if it contained only the two factors A and C, specific for this type, and none of the subsidiary factors. According to the author, the solution so far adopted of preparing vaccine from strains isolated during the epidemic against which control measures are being taken lacks precision. If the overriding necessity of having a completely polyvalent vaccine in stock, comprising the 13 O factors, is admitted, it would seem logical to take into account the antigenic composition of the vibrios responsible either for endemic cases or a specified epidemic. Moreover, it would be advisable to make a selection from those strains containing all the required factors, by quantitative titration of the total O antigenic content of each one, thereby ensuring the production of a vaccine with maximum specific immunizing properties.

nie. Om hierdie rede kan die skrywer saamstem met Burrows dat antigeen A die enigste spesifieke antigeen van die egte cholera-vibrio is (en van die El Tor-vibrio wat serologies identiek is). Ondervinding het ook getoon dat faktor A alleen kan bestaan, sodat dit moontlik is om 'n nuwe en vierdie tipe cholera-vibrio te omskryf. Faktore B en C is noodsaaklik aangesien hulle ten opsigte van die tipes Ogawa (B) en Inaba (C) spesifiek is, maar slegs op 'n ondergeskikte wyse omrede van hulle verhouding tot faktor A. Faktore B en C is trouens meer dikwels in nie-cholera-vibrios teengekom. Faktore D, E, . . . M is van minder belang. Die O-antigeen-formules van die bindbare vibrios en die veelvuldigheid van O-antigeenfaktore in albei groepe vibrios word in twee tabelle saamgevat.

Hierdie proefnemings kan belangrike praktiese gevolge hê: (*a*) In diagnose sal anti-O-monospesifieke serum A in die toekoms eerder gebruik moet word dan Ogawa- en Inaba-serums wat berei word van soorte waarvan die antigeenformule slegs gedeeltelik bekend is. Verklomping weens nie-spesifieke faktore sal dus vermy word. (*b*) Volledige antigeniese ontleding van die cholera-vibrio kan huidige opvattings van die bereiding van entstowwe verander. Ook in hierdie verband skyn die aanvaarde opvatting van die tipes Ogawa, Inaba en Hikojima nou verouderd te wees. Indien die nood ontstaan om 'n sogenoemde eenwaardige entstof te gebruik vir die bestryding van 'n epidemie van 'n vermoedelike ‚enkele' serologiese tipe, sou dit raadsaam wees om met die grootste moontlike sorg te werk te gaan en nie te veel staat te maak op die respektiewe monospesifisiteit van die drie klassieke tipes nie.

Uit antigeniese ontleding het geblyk dat cholera-vibrios van die Egiptiese epidemie van 1947, byna almal van die tipe Inaba, al die faktore behalwe B, G en J bevat het, met A, C, D, E en L as oorheersende faktore. Om hierdie rede is dr. Gallut van mening dat 'n gemengde entstof wat uit een Ogawa- en een Inaba-soort samegestel is nie net onvoldoende blyk nie, maar dat 'n entstof wat net uit die Inaba-tipe saamgestel is gedeeltelik ondoeltreffend mag wees indien dit net die twee faktore A en C bevat wat ten opsigte van hierdie tipe spesifiek is en nie een van die ondergeskikte faktore nie. Volgens die skrywer ontbreek akkuraatheid by die oplossing wat tot dusver aanvaar is deur entstof te berei van soorte wat afgeskei is gedurende die epidemie wat bestry word. As die oorheersende noodsaaklikheid daarvan erken word om 'n volkome veelwaardige entstof in voorraad te hê wat uit die 13 O-faktore bestaan, skyn dit logies te wees om die antigeniese samestelling van die vibrios wat òf vir endemiese gevalle òf vir 'n spesifieke epidemie verantwoordelik is, in aanmerking te neem. Dit sou ook raadsaam wees om 'n keuse te maak uit die soorte wat al die vereiste faktore bevat, deur kwantitatiewe titrering van die gehele O-antigeeninhoud van elkeen, waardeur 'n entstof met die maksimum spesifieke immuniseringsvermoë verkry kan word.

# MALNUTRITIONAL KERATOCONJUNCTIVITIS

## A DISEASE OF THE SOUTH AFRICAN BANTU

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In this article I attempt to summarize the results of 10 years of clinical study of an eye condition which has not, I believe, been previously recognized in this country. For that matter, I am doubtful whether this condition occurs in other countries in quite the same form as in the African, and may, like carcinoma of the liver, be peculiar to his race.

When I left London 10 years ago to start practice in Johannesburg, I had seen none of the conditions which I am about to describe, and it was with some mystification that I regarded, soon after my arrival, my first case of a 'clean' (spontaneous) iris prolapse in a 6-month-old Bantu infant. The parents were quite definite that there had been no injury. On the other hand, I felt equally certain that I had never seen a more traumatic-looking corneal perforating wound with its clean musbroom-like prolapse of iris. However, I abscised the prolapse, and left it at that, but not without a degree of that mental disquiet which accompanies an unsolved problem.

Many months passed before I saw an almost exactly similar case, but in the meanwhile I had come across a series of nearly similar and overlapping corneal conditions which were at first extremely puzzling, until the idea finally crystallized in my mind that I was perhaps witnessing the physical manifestations of a vitally important (and perhaps new and increasing) sociological disease. My eye experience in London had been not inconsiderable (4½ years of concentrated clinical eye work at Moorfields and Guy's), the 'turnover' of patients at Moorfields in those days being 800 daily; nor was malnutrition unknown, particularly when one remembers that both these institutions served some of London's worst slum areas. Although one saw many recognized eye manifestations of malnutrition, e.g. infections of the conjunctiva, corneal ulceration of phlyctenular and other well-known types, the condition which I have designated 'malnutritional keratitis' was conspicuous by its absence.

It may well be that this condition occurs in countries like India and China where large populations live on an almost exclusively starch diet and suffer from a more or less chronic avitaminosis, but I have not yet seen a verbal description or photographic likeness which adequately matches the clinical appearance of 'malnutritional keratitis' as I have seen it in the South African Bantu.

Duke Elder in his encyclopaedic four-volume *Text Book of Ophthalmology* mentions only the xerosis of the older writers who ascribed the disease to lack of vitamin A. The description of xerosis is fairly cursory, and does not fit that of 'malnutritional keratitis'. Moreover, my experience of the latter disease is that lack of vitamins B and C, more especially B, is the causative factor. The word xerosis and its clinical description suggest a drying and desiccation of the cornea, whereas in malnutritional keratitis 'wetness' is more the keynote of the condition, e.g. increase of vascularity, lachrymation, mucus ('wet' catarrh) liquefaction and softening of the cornea ('wet' ulceration), etc. It would appear therefore that malnutritional keratitis and xerosis are two separate clinical entities.

A few years later I was fortunate to get into touch with Mr. Wentworth, the energetic Director of the South African National Council for the Blind. I mentioned to him some of my ideas on the subject and found him a very ready listener. He has in his time, amongst other things, been a practical farmer—and we found to our mutual pleasure that our minds had for some years been thinking on parallel lines, the cornerstone of our views being the importance of soil fertility to health, its conservation and the education and instruction of all South Africans, both black and white, in the proper husbandry of the soil.

This meeting gave birth to the first of a series of nutritional surveys—excellently conducted by him under the auspices of the National Council throughout the Union—which we made in the King William's Town area. At first there was quite a considerable amount of antagonism to our views and there probably still is; I, on the other hand, have found much to quarrel with in the statistics of some of the surveys conducted by other ophthalmologists. Loose classifications of eye disease such as 'conjunctivitis', 'corneal scars', etc., are of little or no value. The misdiagnosis of 'trachoma' for one of the forms of chronic malnutritional keratoconjunctivitis is common. Like trachoma, chronic malnutritional keratoconjunctivitis is a catarrhal state sometimes prolonged over many years, and might therefore be expected to produce a late clinical picture very similar in appearance. There are very few chronic catarrhal conditions of the ocular conjunctiva, and none of the others produce chronic fibrotic changes anything like these two. Spring catarrh with its characteristic symptoms, for instance, can be recognized at a glance and rarely produces seriously damaging permanent changes. The granulomata of tuberculosis, embedded foreign bodies, etc. are rare. Submucosal overgrowth of the yellow elastic components as in the common pterygium has its own clinical picture and cannot be confused with these two. One could continue in the same vein about other chronic ocular diseases, but in no instance could a *common* condition be instanced which produces the same clinical picture as the two above.

There is much in common between trachomatous

and malnutritional catarrh. I have long suspected that trachoma itself is a disease related much more to malnutritional factors than to bad hygiene, dirty surroundings and the rest. There is little doubt that a virus is present; also that chronic secondary infection plays its part (perhaps a more important part than the virus itself), but trachoma is a disease more of the *poor* than of the *dirty*, exactly as is malnutritional keratitis.. It is a serious matter to make a diagnosis of trachoma purely on the evidence of a pannus and corneal scarring similar to (but not the same as) that of trachoma; or on the conjunctival fibrosis which leads eventually to shortening and contracture of the fornices and lids, with the inevitable sequel of inturning lids (entropion and trichiasis). Much of the corneal scarring of late trachoma is produced by the mechanically abrasive effects on the cornea of the inturning lashes and masks the earlier scarring produced by the virus itself. Add to this yet further secondary infection, and little wonder that the two conditions in the late stages mimic each other so closely. To this form of malnutritional (conjunctivo-) keratitis I have given the name of ' pseudo-trachoma '.

In spite of some opposition, however, I am pleased to see that our efforts have convinced some of my colleagues; and the fact remains that if this series of surveys proved nothing else it at least linked up eye disease with:

i. Poor nutrition;

ii. A rural population backward in their knowledge of soil husbandry;

iii. Soil erosion and the like.

Far from being a rare disease, malnutritional keratitis is in my opinion *by far the commonest cause of preventable blindness in South Africa*, and ranks numerically equal with any other of the important causes of blindness, e.g. cataract, glaucoma, industrial trauma, etc. It is important, too, to realize that whereas these latter are diseases of mainly the senile and later age groups, malnutritional blindness is chiefly a disease of the young. Large numbers of these blinded children and young adults, often synchronously afflicted with the other debilitating diseases of malnutrition (e.g. tuberculosis, beri-beri, etc.) and with the mental apathy which runs *pari passu* with a low nutritional level, are no fit subjects for the labour market. Having thus far lived a useless life, they accordingly disappear back to the kraal where, having no earning capacity, they succumb to an early and useless death, thereby serving the double purpose of saving the statisticians much trouble, and trimming down our national figures for blindness, etc.

Malnutritional keratitis is related to nutrition purely in the qualitative and not the quantitative sense. *I have not seen it occur in a really emaciated Bantu child*, i.e. it does not occur as the result of an empty belly. Most of the children are fat and podgy and probably with a white skin would look like the ' watery ', ' carbohydrate ' or ' catarrhal ' child the pediatricians describe.

These are the children said to win prizes at baby shows, a fact which makes the condition all the more sinister. The baby that is ' skin and bone ' and looks ' starved ' in the lay sense, and except for general weakness may be quite fit organically, brings a ready gasp of horror to lay lips. But the baby that looks fat and fit, and may have its eyes and lungs slowly rotting away, is treated as normal even by its parents until a late and often hopeless stage. One is forced to the conclusion, therefore, *that a predominantly carbohydrate diet without vitamins is actually dangerous*, and that if there must be malnutrition in this land of potential plenty *it would almost be preferable to starve the child in the literal sense on a minimal subsistence, but balanced, diet.*

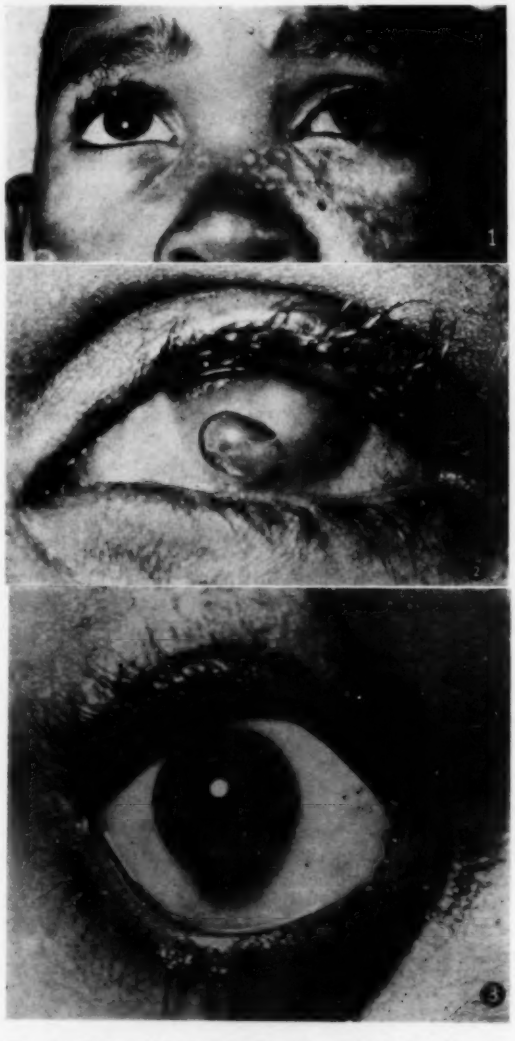


*Fig.* 1. (Type 1.) Left eye: Spontaneous 'clean' iris prolapse actually in process of development at 7 o'clock in a child rather older than usual.

A thin skin of ballooned cornea still covers the prolapse (Descement's endothelium, whole corneal thickness?) and gives it an unnatural shine.

*Fig.* 2. Enlargement of Fig. 1.

*Fig.* 3. A later stage of the same type with more infiltration of the corneal margin around the prolapse.

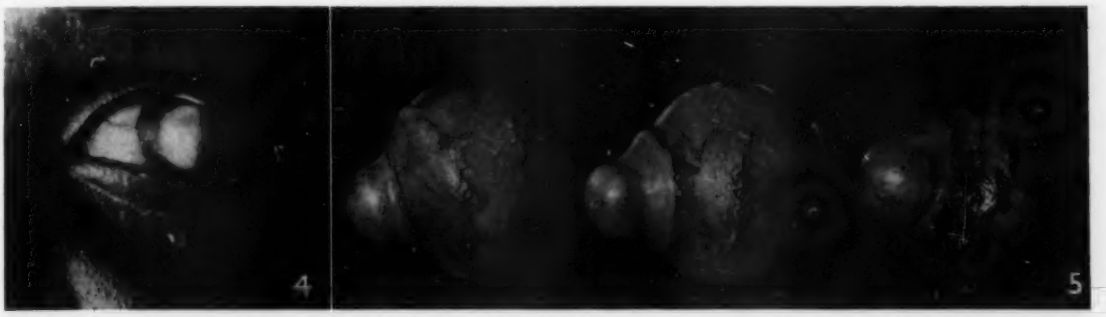


*Fig.* 4. (Type 2). Nipple or teat type.
*Fig.* 5. Excised eyes of nipple or teat type. Note the length of the anterior-posterior axis of the cornea, the constricted neck and the rounded head.

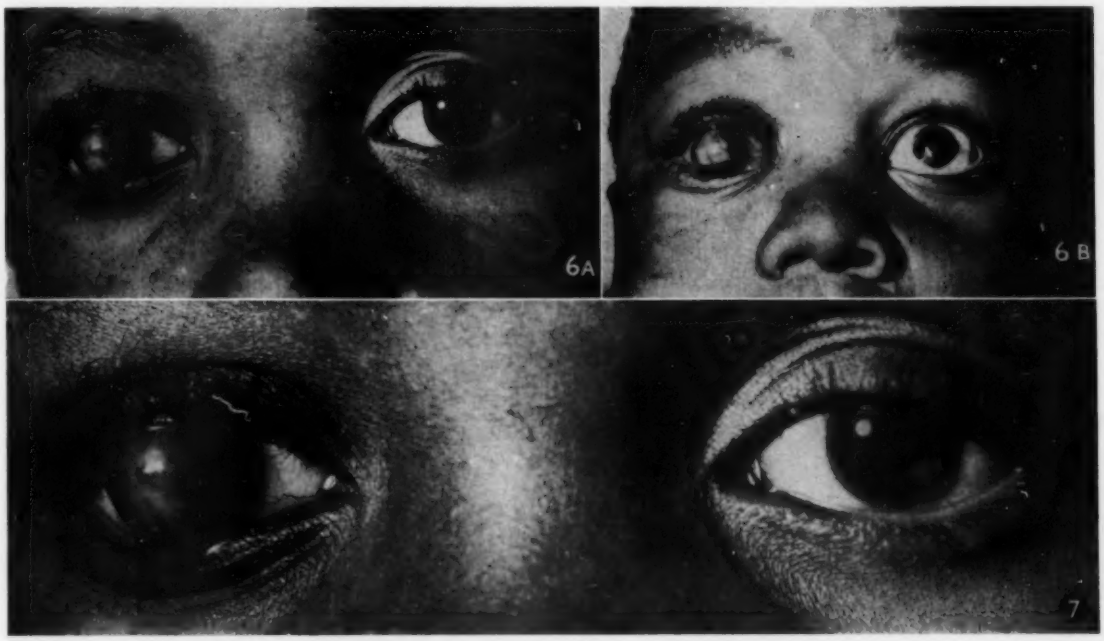


*Fig.* 6a. Type 3 and 4 in the same person—an infant.
*Fig.* 6b. Type 3 and 4 in a much older child (pseudo-buphthalmic right eye; iris adherens left eye).
*Fig.* 7. Enlargement of Fig. 6a.

In this respect the Government school feeding plan with its balanced ration had enormous value. Time will prove what a short-sighted move it was to curtail it. The costly budget for increased blind pensions and other disability allowances, and the cost to the country in diseased and crippled beings, must soon become evident.

The dramatic change in the early and treatable forms of the disease by the exhibition of mixed vitamins is quite remarkable. From a listless, apathetic, photophobic, lachrymating, mewling and miserable creature, the child is transformed in a few days into a cheerful, energetic and happy personality. The dull dry skin recovers its normal shine, the dead lack-lustre hair acquires crispness and sheen, the running nose and eyes dry up, and light becomes tolerable. Most dramatic of all, however, are the changes in the eyes. Quite a hopeless looking cornea, which may even have perforated, responds in young cases in the same extraordinary manner, and confounds the gloomy prognostications of the surgeon. It is difficult to do controlled experiments on human beings, as there are so many variables, but the impression is very strong that with a purely carbohydrate diet the necessity for vitamin B

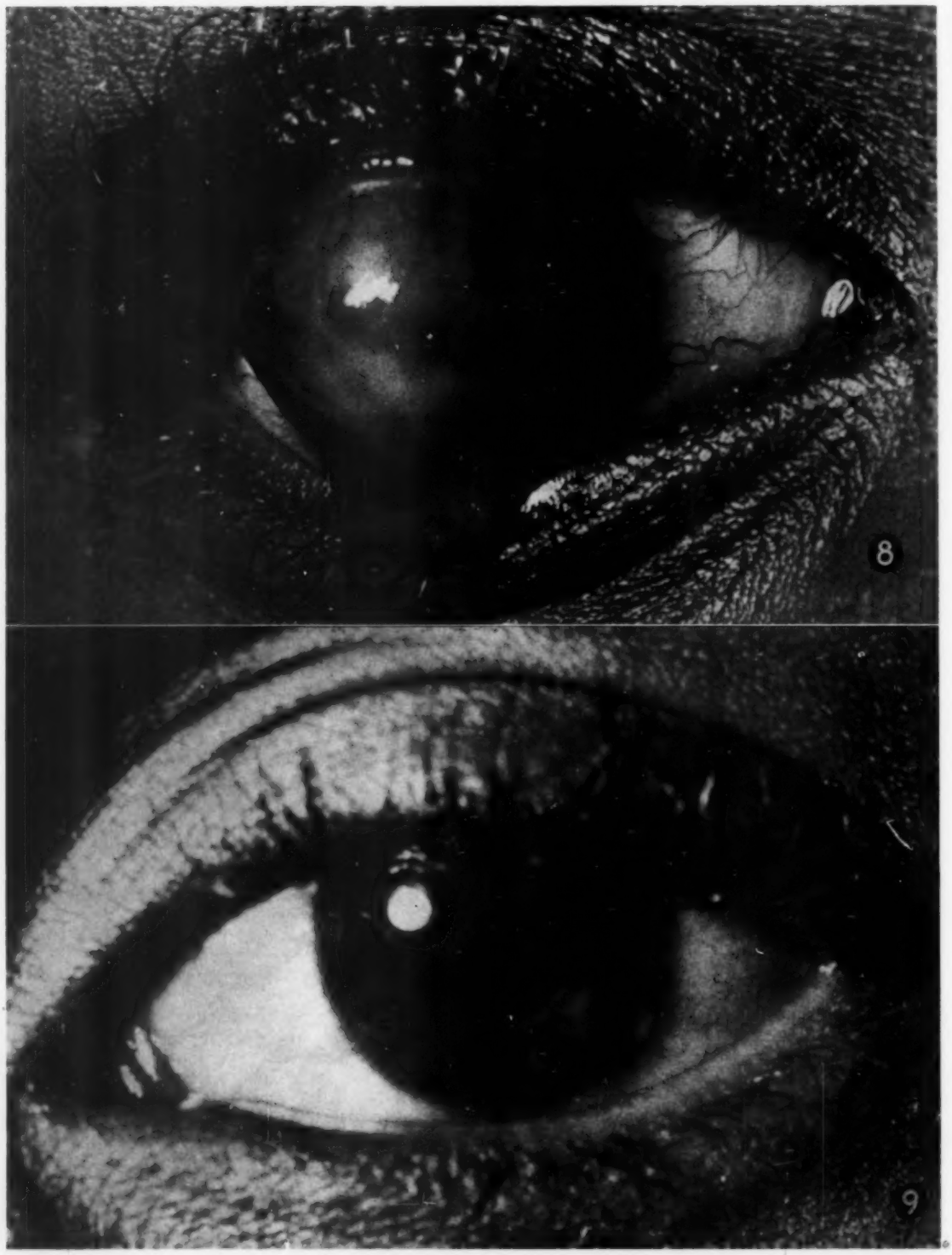
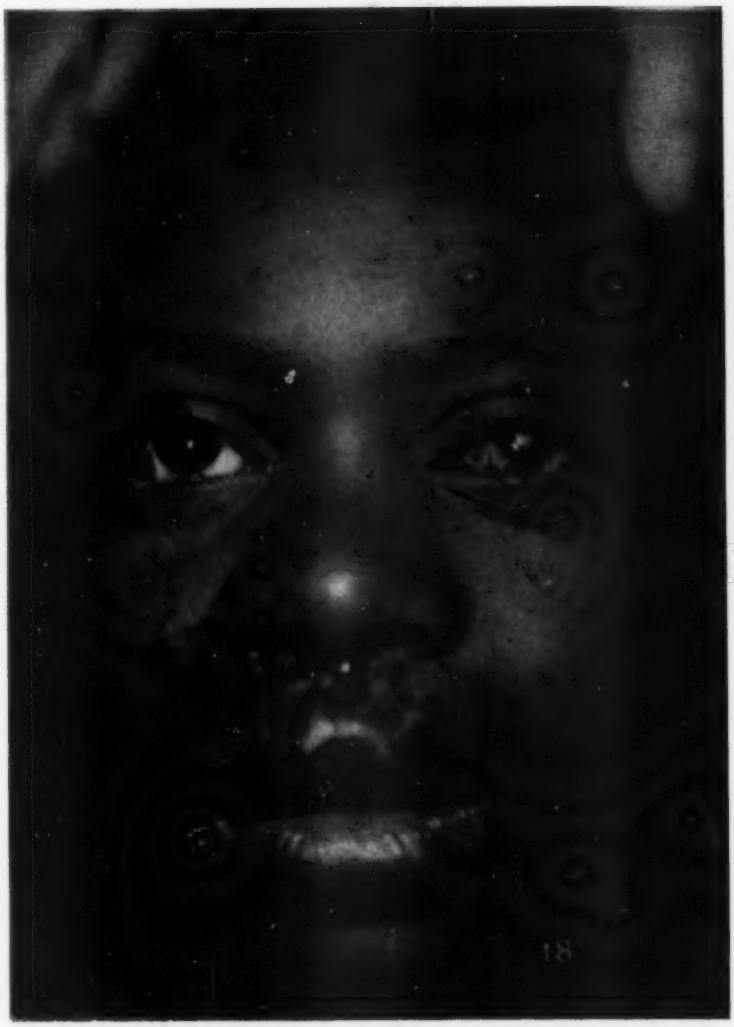



*Fig.* 8. (Type 3). Enlargement of right eye in Fig. 6a.
*Fig.* 9. (Type 4). Enlargement of left eye in Fig. 6a. Note faintness of nebula and shape of pupil indicating iris prolapse.



Type 6. Acute catarrhal stage. Secondary infection here dominates the picture and masks the underlying malnutritional process. Old scars on the left cornea. This form can assume epidemic proportions in badly nourished communities and can leave the eyes scarred and blind within a few weeks.

stands paramount. Local treatment to the eye is quite unnecessary unless a perforation or prolapse is threatening, when atropine should be instilled and the eye bandaged. Antiseptics and irritant drops are positively harmful (as indeed they are in nearly all eye conditions), and more eyes are probably lost by empirical over-treatment than by the much (medically) criticized ministrations of the witch-doctor. The building of mammoth eye hospitals and the outlay of enormous sums for mobile ambulance units for rural areas, although helpful, are not the true answer to the problem. 'The cure of blindness in Southern Africa can only be brought about by its prevention.'

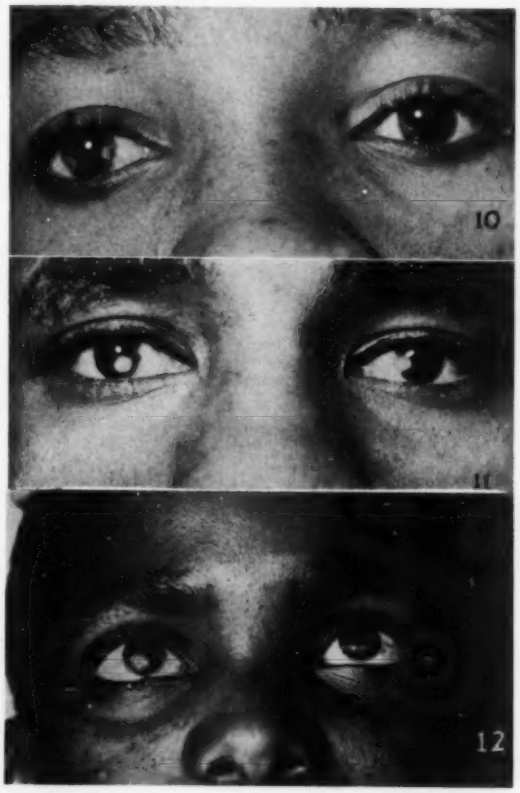



*Fig* 10. (Type 4). Multiple scars with iris adherens or old perforations and prolapse.
*Fig*. 11. Another example of Fig. 10.
*Fig*. 12. Another example of Fig. 10.

## CLINICAL TYPES

### A. CORNEAL SOFTENING OR LIQUEFACTION (WITHOUT SECONDARY INFECTION)

*Type* 1. *Spontaneous or 'Clean' Prolapse (Figs.* 1, 2 *and* 3). I give this condition pride of place, firstly, because clinically it is unique that cornea should dissolve away quietly and insidiously at one small point, without a sign in the eye to attract the superficial attention of the parents or lay observer; and secondly, because it represents the malnutritive process in its pure and unadulterated form. The usual point of corneal dissolution is in the lower segment (either down and in, or down and out).

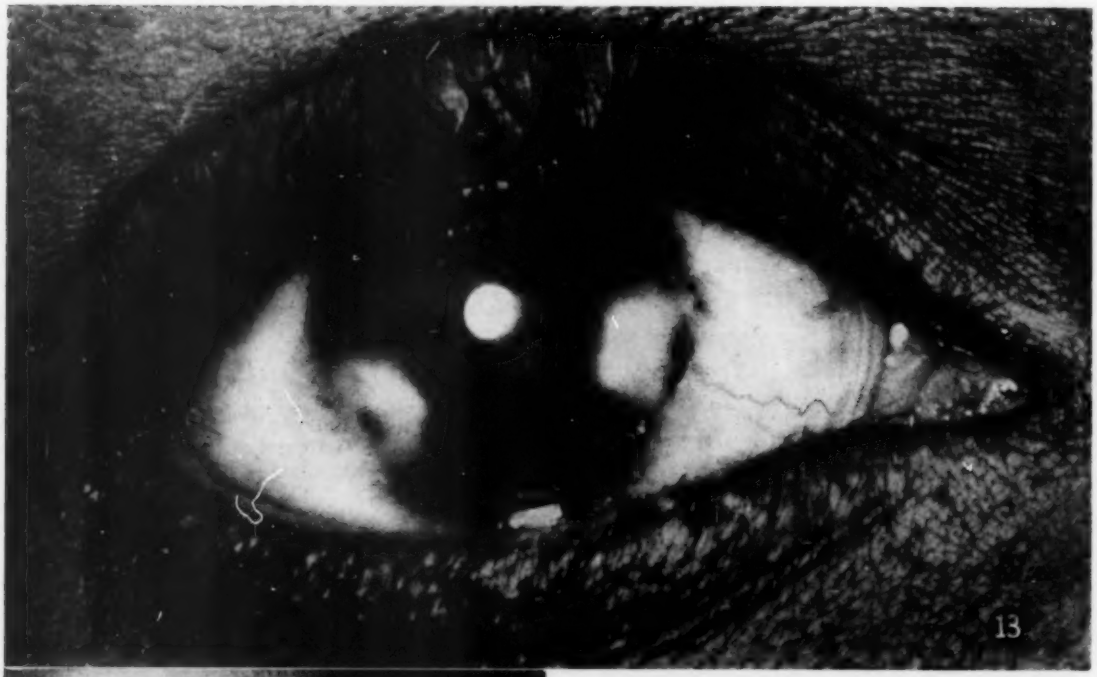
The prolapsed knuckle of iris is usually perfectly round, much like a button mushroom in shape, 1-2 mm. in diameter, and about 1 mm. from the limbus. The eye is nearly always white and uninflamed with not even the suggestion of a cilary flush. I have seen this condition in its pure form only in infants and young children, and rarely after the age of eight. Apparently the eye feels no pain. The mother's attention is drawn to the eye by the presence of 'something black on the eye' rather than by any signs of discomfort, or complaint by the child. Vision is usually little affected, as the only immediate effect is a drawing down of the normally round pupil towards the hole as the aqueous escapes and the iris flows into and finally plugs the leak. This results in the usual elliptical or slit-like-pupil of any iris prolapse. Later, organization of the cornea at the site of the prolapse may produce a moderate degree of astigmatic error.

It is worth recording that in many hundreds of cases of this and other types of malnutritional keratitis I have not seen a single case of iris prolapse in which the other eye was affected by sympathetic ophthalmitis. Indeed, iritis of any sort is uncommon in any of the conditions of this series, and traumatic sympathetic iritis appears to be rare in the Bantu.

*Type* 2. *'Nipple' or 'Teat' Type* (*Figs.* 4 *and* 5). *Corneal Softening with Expansion and Hypertrophic Thickening.* Expansion takes place in the central corneal area in a way similar to one form of conical cornea, leaving a peripherally unaffected margin. There is, however, instead of a cone, a round 'head' and a constricted 'neck', the appearance suggesting the teat of a baby's feeding bottle. The protrusion is often enormous, sometimes as much as a third of an inch outside the lids, closure of which becomes difficult. There is little danger of spontaneous rupture in this eye because of the interesting nature of the pathology.

Section of one of these corneae confirms what is fairly obvious to the naked eye, viz. that two processes are taking place at the same time—one of expansion, the other of hypertrophic thickening of the connective tissue stroma of the substantia propria. The whole 'head' has an ivory whiteness. There is seldom ulceration or infection even in the advanced stages.

*Type* 3. *'Pseudo-Buphthalmic' Type* (*General Corneal Softening with Expansion and Attenuation.* Remarkably enough, this type does not appear in early infancy, but from the first year up to and just after puberty in increasing numbers, so that any suspicion of a glaucomatous aetiology can be discounted. In Figs. 6a, 6b and Fig. 7, I was fortunate to get photographs of the pseudo-buphthalmic condition in one eye (the right eye in each case) and malnutritional ulceration and prolapse in the other. In Fig. 6b the old scar and prolapse is seen at 7 o'clock; and in Figs. 6a and 7 showing more as a nebula at 4 o'clock. Fig. 7 is a close-up of Fig. 6a and Fig. 8 an enlargement of the pseudo-buphthalmic eye of the same patient. Here



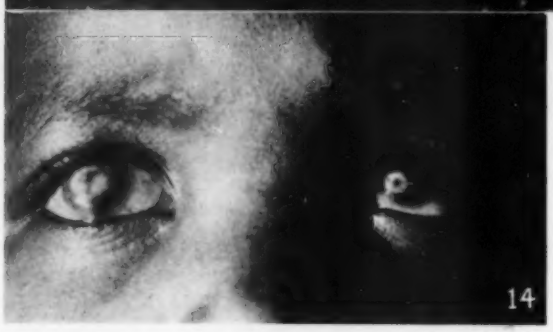




*Fig.* 13. (Type 4). Double perforation and incarceration of iris. Note the shape of the pupil.
*Fig.* 14. Right eye shows acute infection and rupture of old scar with panophthalmitis.

attenuation of the whole cornea to a cellophane thinness is found. The iris usually shares in the stretching and appears to be plastered to the posterior corneal surface. Rupture and panophthalmitis are common (Fig. 14 right eye; left eye, old malnutritional scar and prolapse).

B. CORNEAL SOFTENING AND LIQUEFACTION WITH SECONDARY INFECTION AS THE DOMINATING INFLUENCE

*Type* 4. *Infected Perforating Ulcer with Prolapse and Iris Adherens.* I can bring forward several cogent reasons for putting this type in a separate group from Type 1. It is seen at all ages and may in fact be a late stage of Type 1, but as my classification is clinical and not pathological, and is intended to help those sufficiently interested to recognize them at sight, I make no excuse for doing so.

This is by far the commonest type and there must be many thousands of these eyes in South Africa. An out-patient clinic seldom fails to produce one or two of these per session. As in Type 1, the position of the iris adherens is constant in the lower segment of the cornea. The surrounding scar varies from a marble white density (Fig. 11) to a faint nebula (Fig. 7 left eye) and even in the case of the latter an iris adherens is usually present.

These eyes are usually ' quiet ' but acute exacerbatious of a catarrhal nature are sometimes superimposed on other parts of the eye in younger subjects. Less commonly one sees more than one perforation in the same eye. Fig. 13 and Fig. 10 (right eye) show a good example of a double perforation with the iris incarcerated in both scars. The pupil has become a horizontal slit. I have seen a threefold perforation with iris adherens, but the eye was too scarred to make a good photograph. These multiple perforations with prolapse probably take place at the same time during the same attack. Figs. 11 and 12 show two further examples of this very common type.

*Type* 5. *Gross Central Scarring with Hypertrophic Thickening of the Cornea* (Fig. 16). No perforation; no corneal ballooning. The only means of making certain that one is not dealing with an old gonococcal ophthalmia is to get a careful history from the parents. A point against a gonococcal aetiology is the *absence of perforation,* apparently prevented by the hypertrophic tendency. I attempted a corneal graft later on the

child shown in Fig. 16. The disc of cornea removed at operation showed a striking naked-eye increase of thickness, being nearly double the normal. Microscopic section showed 'thickened stratified epithelium. The substantia propria shows vascularization and hyaline degeneration. There is a thickening of the laminations and an increase of corneal intralamellar connective tissue'.

It was obvious at operation that the donor graft stood a poor chance of survival in its new bed of dense scar. Surprisingly the graft took well, but rapidly became opaque.

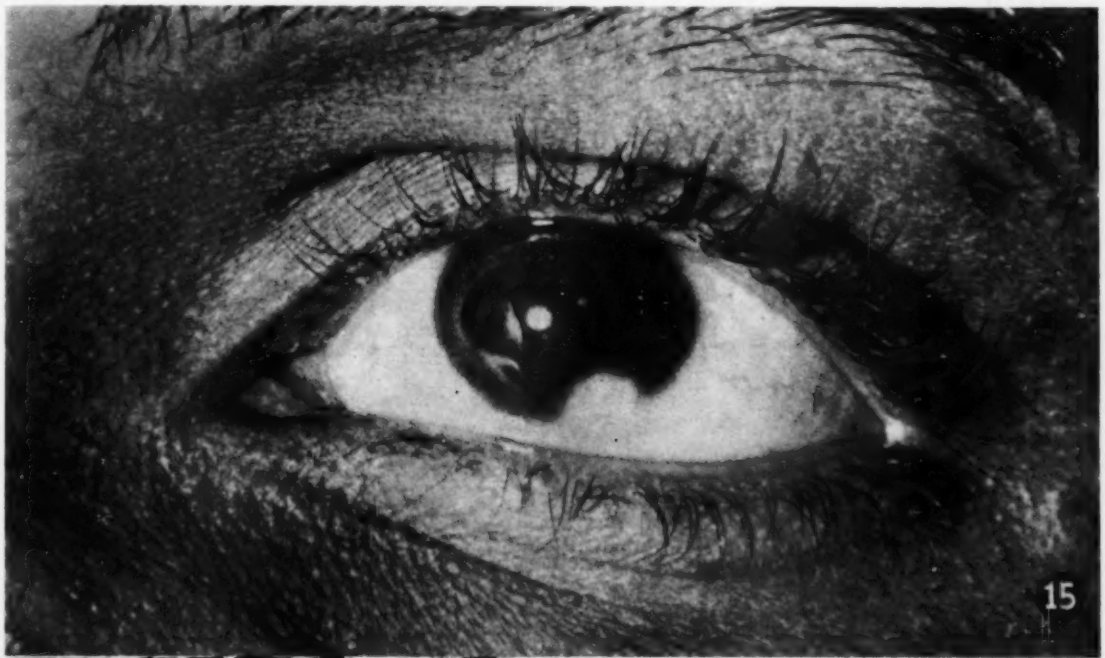



*Fig.* 15. Old scar and perforation with iris adherens.
*Fig.* 16. (Type 5). Central scarring with hypertrophic thickening and relatively little stretching of the cornea.
*Fig.* 17. Pseudo-trachoma (Intermediate Stage). Chronic catarrh with acute exacerbation. Note early evidence of entropion of all lids, especially lower left lid where lashes can just be seen to be assuming vertical as opposed to normal horizontal position. Trichiasis and abrasion of the cornea by the inturning lashes is not far off. Palpebral apertures smaller than usual from contracture (fibrotic) of subconjunctival and deeper lid substance. Corneal scarring and pseudo-pannus are a marked feature.

### C. CATARRHAL KERATOCONJUNCTIVITIS

*Type 6. Acute Form.* Excessive lachrymation, increased mucus, a little pus and severe photophobia are typical of this condition Impetigo of the face, nose or ears is sometimes present. The child is miserable and uncooperative. Loss of corneal sheen is a warning of threatening fulminating ulceration. This is the type that reacts dramatically to good food and vitamins. Naturally one cannot be certain as to which of the previously mentioned forms the condition will develop into, but my surmise is that it would be the common form (Type 4). This type is difficult to photograph with a still camera (Fig. 18) but is well illustrated by cine film. One often sees this acute form as an exacerbation on the others.

*Type 7. Pseudo-Trachomatous Malnutritional Kerato-Conjunctivitis.* I have already dealt with this condition at some length earlier in this article. The

ultimate clinical picture is seen mostly in the older Bantu and is *almost indistinguishable from a case of old trachoma*, but I believe there are differences. The condition results from recurrent low-grade attacks of the acute form (Type 6) acting over many years. Acute and sub-acute trachoma on the other hand can be quite easily distinguished from this type and are, in my opinion, relatively uncommon in this country. The number of Type 7 'pseudo trachomatous' cases that show themselves at clinics is quite out of proportion to the number of cases of 'fresh' (acute and sub-acute) trachoma seen, and are nearly always senile Africans almost totally blind from the effects of the long-continued trichiasis on the cornea. This type also does not photograph well.

SUMMARY

A new condition of the eye is described as seen through the eyes of the writer over a period of 10 years.

The condition is directly linked to those evils which threaten the immediate future of the people of this country, namely, loss of soil fertility, soil erosion, sloth, ignorance, indifference towards and the abuse of the land. Lack of quality in food is more important in producing malnutritional changes than lack of quantity.

Malnutritional blindness is numerically the only important cause of preventable blindness in the Union and affects the young. Other forms of blindness (non-preventable) occur mainly in the old, senile and less useful members of the population.

A description of the writer's seven clinical types is given.

I would like to thank Mr. Marius Garb for developing and enlarging the photographs and for his valuable suggestions; my house surgeon, Dr. Chait, for his help in dealing so patiently with the difficult temperaments of patients, camera and chief alike.

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# MYANESIN IN THE BANTU*

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We have used Myanesin in this hospital for 293 cases. The ages of our patients have ranged from five hours to 65 years, and their condition has been from good to serious.

A non-European hospital, where the majority of cases are Bantu, presents a different anaesthetic problem in comparison with a European hospital.

The Bantu, more particularly the urban Bantu, suffers from the effects of malnutrition. He generally has an avitaminosis, particularly of the B and C series, and Gillman and Gillman have drawn attention to the fact that there is a high incidence of severe liver disease in presumably healthy Bantus in Johannesburg.

Deaths under anaesthesia among the Bantu appear to be higher than among the Europeans, owing to the type of case which arrives at operation.

The Bantu is also more difficult to anaesthetise. Dill states that Negroes and children are particularly susceptible to apprehension, fright and panic, and shows that in a series of 10,580 anaesthesias, during five years, there were five deaths, all among negroes.

The Bantu goes into spasm more easily than the European, has many loose and septic teeth, generally has marked prognathism and, above all, is usually so dark in colour that 'cyanosis may not easily be detected' (Lundy).

The cases in this series may be divided into the following categories:

* This paper was read at the Medical Congress of the Association held at Cape Town in September 1949.

*Orthopaedic* (17 *Cases*). A vast amount of the work done in a hospital which caters solely for the non-European is orthopaedic work. In these cases Myanesin was used only as an aid to intubation.

*Gynaecological Cases*. In the series presented here, 88 were abdominal gynaecological cases. These women, who are often obese, have generally been in a state of chronic ill-health for a number of years. On admission to hospital, their haemoglobin is often very low, and they may need one to two transfusions pre-operatively, in order to bring it to the 65% to 70% which appears to be the lowest level suitable for a non-emergency abdominal operation.

*Cold Surgical Cases* (*other than Orthopaedic or Gynaecological*). Eighty-six cases were in this category. Of these, 21 were upper abdominals. The Bantu usually waits a long time before coming to hospital. Thus, by the time he comes to the operating theatre, he has a history of long standing and is, in about 50% of cases, a poor operative risk.

*Emergencies*, which constitute 87 of the cases. These emergencies may be divided into:

*a. Acute Abdomens*, totalling 33 of the total emergencies; the majority being intestinal obstructions and extremely toxic.

*b. Disembowelments*, numbering 49, and

*c. Bullet Wounds*, numbering 5.

*Cases where, owing to the Site of the Operation, Intubation was necessary*. These comprise fractured jaws and head cases, where the Myanesin was used as an aid to intubation, a total of 15.

It is not uncommon, in a disembowelment, to find a patient with six or more perforations into the bowel. As a rule these cases are in a state of profound shock. The majority have stomachs full of kaffir beer or skokiaan mixed with thick porridge and lumps of meat. Where possible, stomach wash-outs are done before the patient comes to the theatre, but this is impossible if there is a likelihood of bowel perforation, and all that can be done to the latter type of case is to insert a Ryle's tube. It is in this type of case that we have found Myanesin of particular value.

The following case is illustrative of the type of emergency discussed above.

A young Bantu male, aged 24 years, was admitted as a disembowelment. His condition was serious, hist respiration gasping, and he appeared to be bled out. His blood pressure on admission was 90/50 mm. Hg. He was treated for shock and given blood intravenously. Two hours after admission his blood pressure had risen to 118/86 mm. Hg. His breathing was still gasping and he was very restless.

Following on premedication with atropine 1/50 and Omnipon gr. 1/3, he was induced with .35 gram Thiopentone, followed by 10 c.c. Myanesin, and then maintained under nitrous oxide, oxygen and a minimum of ether. He was intubated orally and the tube packed off.

At operation, 13 perforations of the small bowel and mesentary were sutured.

Throughout the operation, which lasted two and a quarter hours, his breathing was gasping and respiration had to be assisted. His blood pressure fell, ten minutes after the operation commenced, to 56 mm. Hg. systolic, the diastolic being unobtainable.

The blood pressure remained very low and at the end of the operation was 70/40 mm. Hg. His pulse remained between 140 and 120.

He had no post-operative complications, and was discharged from hospital fifteen days later.

Myanesin, as with d-tubocurarine, may be used in any combination of anaesthetic. In this series we have used it with nitrous oxide, oxygen and ether; nitrous oxide, oxygen and trilene; cyclopropane; nitrous oxide, oxygen and Pentothal; and with Avertin; the majority of cases being in the nitrous oxide, oxygen and ether series.

Our preference for this combination is because we have found from experience that the anaesthetic of choice in badly shocked cases is nitrous oxide and oxygen, the ether being used in minimum quantities, in order to raise the blood pressure. Many of these disembowelments take several hours for operation, and we feel that this is a contra-indication to cyclopropane, in view of cyclo shock developing post-operatively.

As the emergencies are from the ward of intakes, which may be in a congested state with many of the beds holding patients variously injured and often drunk, it is obvious that the patient cannot always be assured of being adequately nursed post-operatively, and it is therefore as well to have his condition as static as possible before sending him back to the ward. This has been found to be most easily obtained with nitrous oxide, oxygen, Myanesin and a trace of ether.

Here it is interesting to note that Dill states that 'the percentage of alcoholic psychosis in the New York State Hospital was highest in the African race'.

The average amount of Myanesin used has been 10 c.c. although in the last 50 cases we have used up to 20 c.c. Not all of our patients were intubated, although intubation with Myanesin is a simple procedure.

The usual technique in cases where Myanesin is used is as follows:

The patients are premedicated with one-third of Omnipon and one-fiftieth of atropine (according to age). They are then induced with either Kemithal or Pentothal (Thiopentone) and as soon as they are unconscious, 5 to 10 c.c. of Myanesin is given them through the same needle. They are then put on to nitrous oxide, oxygen and ether vapour until they are in light second plane. At the end of this period the jaw and vocal cords are relaxed and the patient is intubated orally. If the case is an emergency, a stomach tube may be ntroduced and the tube packed off. The anaesthetic is then continued in light second plane, and if the relaxation does not appear to be sufficient, another 5 to 10 c.c. of Myanesin given.

The relaxation obtained in these cases has been adequate. I have never used the large doses advocated by Cullen, the largest dose being 20 c.c. This is probably why respiration has not been affected at all in the greater majority of cases. This is contrary to the findings of Cullen, but I feel that it is because he used Myanesin with Pentothal that he found it necessary in so many cases to use artificial respiration. On a few occasions, when using Myanesin in combination with Pentothal, artificial respiration has been necessary. I feel that this is due to the synergic action of Pentothal with Myanesin.

### THE USE OF MYANESIN WITH PENTOTHAL AND KEMITHAL

Myanesin has a definite synergic action when used with Kemithal and Pentothal, and used with these drugs has a valuable place in cases where the patient has a history of respiratory disease or where irritation of the respiratory tract has specially to be avoided. In using Pentothal or Kemithal together with Myanesin, relaxation of muscle may be obtained without pushing the Pentothal to dangerous levels. Respiration is more likely to be depressed with this combination of anaesthetic, but I am of the opinion that this depression is due rather to the barbiturate than to the Myanesin. I have not found that the amount of Myanesin that is required when Pentothal is the anaesthetic is any greater than when ether is the anaesthetic.

Myanesin may be used in the same syringe as Pentothal, in the proportion of half a gram of Pentothal to 10 c.c. of Myanesin, and intubation can as a rule be carried out fairly rapidly. It may also, as with d-tubocurarine, be used in a different syringe to the Pentothal, either before the Pentothal is given, or after.

I have used Myanesin in several thyroids, both toxic and non-toxic, as an aid to intubation, following on Avertin (.1 c.c. per kilo body weight). Cases for thyroidectomy in this hospital are often of long standing, and can show marked distortion of the trachea. Giving 10

c.c. of Myanesin, makes intubation easier; d-tubocurarine in these cases would be definitely contra-indicated, owing to the already depressed respiration caused by Avertin.

Myanesin, with Pentothal or Kemithal, is not a good combination to use in badly shocked cases, as the already low blood pressure is kept depressed by the Pentothal, whereas ether raises the blood pressure. This is well illustrated by the following case:

An elderly Bantu, stuporous and smelling strongly of liquor, had to have a compound fracture of his skull elevated and a Roger-Anderson splint applied to his compound-fractured mandible. His condition was serious. His blood pressure was 98/70 mm. Hg.

He was given .8 gram of Kemithal and 10 c.c. of Myanesin into an intravenous drip, and then intubated orally with a laryngoscope. The anaesthetic was continued with 50% nitrous oxide and oxygen, Kemithal being given at intervals when necessary. In all a total of 1.8 grams of Kemithal was given. After three-quarters of an hour his blood pressure, which has been dropping throughout the operation, fell to 0 mm. Hg. He was immediately switched over to a trace of ether, and when he left the theatre, twenty minutes later, his blood pressure was 82/64 mm. Hg.

He died 15 hours later, and at post-mortem was found to have contusions and lacerations of the brain from the orbital area extending up to the parietal area. He also had an adherent pericarditis.

Owing to the flexibility of the dose of Myanesin, this combination with Kemithal (or Pentothal) may be used in the very young or very ill. This has also been found by Davison, and Wilson and Gordon.

The use of Myanesin in the very ill and very young is well illustrated by the following case:

A female Bantu child, aged three years and ten months, was to have a laparotomy, for the removal of a congenital cyst of the liver. The child was in a serious condition. She had a chronic cough, and clinical examination showed decreased air entry at the base of the left lung with rales and rhonchi in the right lung. Radiological examination of the chest showed a pleural effusion of the middle lobe of the left lung. Her haemoglobin was 11.3 grams per cent. and her red blood count was 3,700,000. Premedication was 1/100 atropine three-quarters of an hour pre-operatively. She was induced with 2½% Pentothal, and when unconscious was put on to 5% glucose in normal saline, intravenously. Pentothal, in all .375 of a gram, was injected into the drip as required, together with 3 c.c. of Myanesin. Oxygen was given through a Boyle's machine. The operation took one hour and a quarter, and the patient gave no cause for anxiety throughout. There were no post-operative complications and she left the hospital fit two months later.

This combination of Myanesin and Kemithal was found to be of value in obese patients with chronic coughs, too fat for spinals. The following case will amplify this point:

The patient was a woman, aged 60 years, weighing 350 lb., with an enormous ventral hernia for repair. She had a chronic cough, for which no cause could be found either clinically or radiologically.

She was induced with Kemithal, followed by 12 c.c. of Myanesin. She immediately went into marked spasm, and was therefore given more Kemithal, until in all she had had 3 grams of Kemithal. Intubation under vision was done with great difficulty owing to the shortness of her neck plus her obesity. Her throat was full of mucoid pus, apparently coming from her antrum, this probably being the cause of her chronic cough and spasm.

Once intubated, she was kept on 50% of nitrous oxide and oxygen, and given Kemithal when necessary. Altogether, over four hours and ten minutes, she had 6.4 grams of Kemithal and 17.5 c.c. of Myanesin. During the operation, which was extremely difficult, her condition gave no cause for anxiety. At the end of the operation she was given 40 c.c. of sodium succinate. She was completely conscious 45 minutes after leaving the theatre. Post-operatively she had a chronic cough but was up on the second day, and left hospital one month later fit.

This patient was interesting in that, although she showed a trace of albumin in her urine, she showed no after-effects from the Myanesin.

Myanesin is the ideal relaxant for the badly shocked patient, and since its introduction into this hospital I have felt far less anxiety in dealing with patients who are gravely ill; there is no doubt that many patients have left the operating table alive who would, in all probability, have had small chance of survival if ether had been the relaxant.

This greater margin of safety has also been noted by Marston and Davison.

Myanesin appears to have no effect on cardiac musculature, and I have used it in several cases with a history of either myocarditis, hypertension or of a recent cardiac failure where adequate relaxation was needed.

This is substantiated by the work of Berger which proved that toxic doses of Myanesin caused death by respiratory failure, the heart as a rule continuing to beat after respiration ceased.

The blood pressure and pulse rate and volume showed no significant changes in this series. This is consistent with the findings of Wilson and Gordon and of Ballentine.

A patient who has had Myanesin and who appears to be well relaxed, will, if not analgesic enough, strain and push the abdominal contents down, with his unparalysed diaphragm. Because of this, it is impossible to maintain a patient who has had Myanesin in such a plane of anaesthesia that he can feel stimuli.

In the Bantu, the incidence of chest complications following an abdominal operation is higher than in the European. Following on the use of Myanesin, this incidence would appear to be reduced. This is contrary to the findings of Organe on d-tubocurarine, who reports an unduly large number of serious post-operative chest complications following the use of d-tubocurarine.

In none of our cases have I seen the shock-like state that is reported by many anaesthetists after the use of d-tubocurarine. This shock-like state is held by many

to be due to the patient being relaxed, without being sufficiently analgesic.

All these cases regained consciousness very quickly, and the post-operative and general condition of the patient was excellent.

In this series we have had no deaths attributable to the Myanesin. As many of these cases have been very poor risks, there have been some deaths, days and even weeks later, in the wards, but these deaths have in every instance been due to the primary cause of the illness and not to the Myanesin.

I have noticed no clinical effects on the intestines or increase or decrease in bleeding. Ballantine agrees with this.

Bantu patients seldom vomit post-operatively, so it is difficult to assess what effect Myanesin has on vomiting. Wilson and Gordon state that it is lowered in the European by the use of Myanesin.

There have been a very small percentage of cases of thrombosis following on the use of Myanesin, but I feel that this may be due to the fact that we use the same vein for the Myanesin as we use for Kemithal or Thiopentone.

Mallinson states that he has had no cases of thrombosis as yet, whilst Stephen and Chandy found thrombosis in seven out of fifteen cases. Lyall found thrombosis in one case out of two hundred and fifty. Vartan and Cullen have also reported cases.

I have found that there is some degree of naked-eye haemolysis after the average dose of 10 c.c., lasting for approximately five hours post-operatively. This has also been noted by Lyall and others. We have, however, had no cases of haemoglobinurea or anuria.

Ogilvie, Penfold and Clendon, writing in *The Lancet*, state that they found that when blood and Myanesin are mixed together profound changes take place. In low concentrations there is little or no effect; in higher concentrations haemolysis takes place; and in higher concentrations still, the blood apparently curdles. It may, in fact, turn solid. They have postulated the theory that their case of gangrene, following an intra-arterial injection of Myanesin, may have been due to the profound change which takes place when blood and Myanesin are mixed, the coagula formed being shot into the smaller arterioles, blocking them.

We have had one case of gangrene of the arm, following on the intra- or peri-arterial injection of Kemithal and Myanesin. Here a spasm of the artery followed immediately on the injection, and I feel that the gangrene was primarily caused by the violently irritating effects of the Kemithal.

This case is at present being prepared for publication.

In view of the tendency of Myanesin to cause thrombosis and haemolysis, and since Enderby and Pugh and, more recently, Torrens, Edwards and Wood, have found that in a more dilute solution, these complications do not occur, I feel that wherever possible, the Myanesin should be used in an intravenous drip or diluted.

### A COMPARISON BETWEEN MYANESIN AND D-TUBOCURARINE

Intubation in the Bantu presents a different problem from intubation in the European. Anatomically, the bridge of the Bantu's nose is small and flat, and the use of any intratracheal tube bigger than a No. 5, often causes marked bleeding. The Bantu also appears to have a nasal mucosa which, in at least 50% of cases, haemorrhages when blind intubation is attempted. It is obvious, therefore, that wherever possible, intubation by the nasal route is to be avoided, firstly, because of probable epistaxis, and secondly, because it is ill-advised to use too small an intratracheal tube, more particularly where the pharynx is packed off. Thus, intubation in the Bantu is best done orally with a laryngoscope. Owing to the marked prognathism of the jaw, and the presence often of many loose and septic teeth, adequate relaxation is very definitely required, and we have come to regard the use of Myanesin or d-tubocurarine as a necessity.

For intubation in the healthy patient by an experienced anaesthetist, d-tubocurarine has a marked advantage over Myanesin. The relaxation of the jaw, and exposure of the cords obtained with 15 mg. or more of d-tubocurarine, is more adequate than that obtained with 10 c.c. of Myanesin.

When d-tubocurarine was first advocated for use in shocked emergencies, I began to use it as a routine; but I have found, particularly in cases where the stomach is full of liquor or food, that the extreme relaxation of the cardiac sphincter of the stomach, obtained in a matter of seconds with d-tubocurarine, is a disadvantage. Before one has time to intubate and pack off the larynx, stomach contents are regurgitated.

Lyall states that, following on the use of d-tubocurarine, attempts at artificial respiration by thoracic compression can force gastric contents upwards to the pharynx and through the paralysed glottis to the lungs. Thus, when using d-tubocurarine, artificial respiration should be done with a positive pressure machine, an intratracheal tube should be passed, and the pharynx packed off. This would not appear to be necessary when Myanesin is used. Myanesin may even be used with open ether.

Because of the known side-effects of Myanesin, it is as well never to use more than 20 mg. of the drug. This is one of its greatest disadvantages in comparison with d-tubocurarine, where, in the non-sensitive patient, enormous doses of d-tubocurarine may be given, with a consequent reduction in the amount of anaesthetic agent used.

Lyall feels that in the extremely ill patient d-tubocurarine has a more protective action than Myanesin. I cannot agree with this opinion. In the shocked emergency, in the young, in the very ill, and in any type of case (as, for example, head injuries) where cessation of respiration, however transitory, would jeopardise the chances of the patient, Myanesin, not d-tubocurarine, is the relaxant of choice.

In all other types of cases, the skilled anaesthetist will prefer d-tubocurarine to Myanesin. However, the G.P. anaesthetist, battling with poor equipment in the country, will find that Myanesin, when used judiciously, is a drug with great possibilities as an aid to relaxation and intubation.

### CONCLUSION

The use of Myanesin as a relaxant is reported in 293 cases.

With Myanesin it is possible to get third or fourth plane relaxation whilst maintaining the patient in second plane anaesthesia.

Myanesin may be used with open ether.

Myanesin does not relax the vocal cords to the same extent as d-tubocurarine does.

Myanesin, used with discretion, is an asset to the G.P. anaesthetist and to the junior anaesthetist. The skilled anaesthetist will find d-tubocurarine the more useful drug, except in certain cases such as shocked emergencies, extremes of age, head cases, or anywhere where depression of respiration is of disadvantage to the patient.

Whilst Myanesin has a synergic action when used with Pentothal and the barbiturates, the necessary limitation of dosage is a disadvantage in comparison to the 'Pentothal-Curare' combination.

Because of the tendency of Myanesin to cause thrombosis and fragility of the red blood corpuscles, and because of reported cases of anuria following on it, use, Myanesin should always be used in dilute solutionss preferably into an intravenous drip. Because of these side-effects, it would appear inadvisable to use more than 20 mg.

Myanesin may be added with confidence to the already formidable array of drugs at the command of the anaesthetist.

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## PASSING EVENTS

### BACK NUMBERS OF THE JOURNAL PUBLISHED ON 7 JANUARY 1950

The editorial office requires back numbers of the issue of the *Journal* published on 7 January 1950. It would be appreciated if those members who do not file their copies and who have no further use for this particular issue would be good enough to forward them to the Editor at P.O. Box 643, Cape Town.

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The next meeting of the Cape Town Paediatric Group will be addressed by Dr. I. Schrire on *Dwarfism* in the Small Lecture Theatre, Groote Schuur Hospital on Monday 27 March 1950 at 8 p.m.

## IN MEMORIAM

### DR. WALTER SHANKS

Dr. Walter Shanks passed away suddenly in his surgery at Humansdorp on 9 December 1949.

Born in Dundrum, County Dublin on 14 August 1875, he spent his childhood in Dublin where his father was the Lord Mayor at the time of the Chicago Exhibition.

Educated at Rathmines School, Dublin, he proceeded to Trinity College, graduating M.B. Dublin University gold medallist in 1898, serving his internship at the Rotunda and Sir Patrick Dun's Hospital.

He came to South Africa in 1899 with the R.A.M.C. and served in the Boer War, after which he was with the South African Constabulary until 1902.

In 1901 he married May Adkins of Fort Jackson and after his discharge practiced for a little while at West Bank, East London, then at Kumgha, C.P. until 1911, when he graduated M.D. Dublin University. After serving as locum on the Rand for six months he moved to Molteno, where he acted as District Surgeon until 1921. In 1915 he joined up with the Union Forces and served as Medical Officer with the Ninth and Tenth South African Horse in East Africa and was mentioned in dispatches. In 1918 he returned to private practice in Molteno, proceeding to Humansdorp in 1921 to take over the practice of the late Dr. J. J. Coulton, acting as District Surgeon until a year before his death.

Dr. Shanks is survived by his widow and two boys, Jimmie and Derry, both with their own families. The daughter Sheila, predeceased her father in 1942, leaving the two grandchildren Jimmie and Jennifer to the care of their grandparents.

Mrs. Shanks is settling down in Uitenhage to take care of the grandchildren.

Dr. Shanks was very devoted to his family; he was greatly respected by his colleagues, who along with his friends and patients regret the loss of a highly cultured gentleman and conscientious professional man.

## BOOK REVIEW

### VARICOSE VEINS

*Varicose Veins.* By R. Rowden Foote. (Pp. 225 + xiv. With 181 illustrations and two coloured plates. 37s. 6d.) Butterworth & Co. (Africa), Ltd., 1 Lincoln's Court, Masonic Grove, Durban. 1949.

*Contents:* 1. Some Historical Landmarks in the Treatment of Varicose Veins. 2. Anatomy, Physiology and Pathology. 3. Incidence and Aetiology of Varices. 4. The Investigation of the Patient with Varicose Veins. 5. Treatment. 6. Varicose Ulceration. 7. Thrombophlebitis and Pulmonary Embolism. 8. Anti-Coagulant Therapy. 9. The Supportive and Compression Treatment in Varicose Conditions. 10. The Varicose Vein Clinic and some Useful Prescriptions. 11. Recent Work in Varicose Veins.

This monograph is an exposition of current sclerosing therapy with or without adjuvant operation in the treatment of varicose veins. The presentation of the contents is attractive and the illustrations abundant though not always clear. There are excellent chapters on the high-resection operation in the groin and the differential diagnosis of varicose ulceration. The chapter on supportive and compression bandaging is superb.

The opening chapter, an historical survey obviously the result of much research, discloses many items of interest, e.g., that Rima (1777-1843), an Italian surgeon, performed the first ligation in the thigh for varices (thus anticipating Trendelenburg), and that Trendelenburg published an account of the test that bears his name, 50 years after it had been carefully described by Sir Benjamin Brodie (1783-1862). The treatment of varices by a combination of operation and sclerosant was first employed by Schiassi of Bologna (1909), while the high-resection operation, the basis of modern

operative therapy of varicosities, was first performed by Homans (1916).

In the second chapter, the conventional account of the venous anatomy of the limb is augmented to include the recent work of Daseler *et al.* by an illustrated summary depicting the varied modes of termination of the groin tributaries in the great saphenous and femoral veins. Brief reference is here made to the 'mid-Hunter canal blowout', an important channel connecting the femoral and great saphenous veins just below mid-thigh. The great saphenous is always referred to as a single channel. It is, however, necessary to realize, as Sherman's epochal studies intimate, that several communicators exist between the femoral and saphenous in the distal thigh (the mid-Hunter canal, the subsartorial, and the geniculate plexus perforators) and that these communicators are not single channels but plexuses. Should one pathway be obstructed by a thrombus, the others will successfully maintain their flow to the saphenous while negotiating patency of the affected venule. The great saphenous, moreover, as Sherman has shown, is very frequently of a duplicate or triplicate nature in the thigh, certain of the channels being deep to the deep fascia and others superficial thereto. Hence, if the ureteric catheter or long fine cannula with nutmeg-grater terminal delivering the sclerosant is passed into one channel, will not the other channels be missed?

In refutation of Edwards (1934), the author rightly stresses the necessity for dividing every tributary entering the highest 2 inches of the great saphenous if every avenue for recanalization is to be removed. On p. 25, the lymph node of Cloquet is depicted superficial to the fascia lata, whereas it should be invisible (in the femoral canal). On p. 25 emphasis is laid upon the angle (60°) at which the great saphenous joins the femoral in the groin. This angle is the most reliable means of identifying the saphenous and distinguishing it from the femoral. The third chapter discusses the etiology of varices and is full of interest. The research on the paucity or absence of valves in the external iliac and upper femoral veins, though attributed to Sherman and Caspar, was actually done by Eger, S. A. and Caspar, S. L. (1943): J. Amer. Med. Assoc., **123**, 149.

Chapter 4 stresses the importance of the general examination, and especially where pain is the complaint, noting the presence of swollen knees and flat feet. Inspection and palpation of the limb are sufficient to indicate the treatment required. Complicated venous competency tests are not recommended, while phlebography is considered of little avail. In Chapter 5, the author states: 'There is no entirely satisfactory method . . . causing permanent eradications of varices'. This view is erroneous; it is born of too great reliance on sclerosants. The three chief penalties after the use of sclerosants are: a high recurrence rate (57% within one year, after the high-resection operation and instillation of sclerosants, according to Tuomikoski (excerpt J. Amer. Med. Assoc., 26 March, 1949); the frequency and peril of deep phlebitis (warnings by Sir Heneage Ogilvie, Boyd, Hanschell, Atlas and others); and fatal pulmonary embolism (Vaughn in 1944 culled from the literature 44 deaths after the use of sclerosants, and added one of his own).

Despite the author's preference for treating post-deep-phlebitic ulceration by support and compression only, dramatic results have, in the reviewer's experience, followed division of the popliteal vein (Gunnar Bauer's operation) aided in the presence of vasospasm (cyanosed, cold and sweating limb, and characteristic changes in skin temperature after reflex heating) by lumbar sympathectomy.

## CORRESPONDENCE

### FALSE MEDICAL BELIEFS

*To the Editor:* Experienced medical men have long come to realize the importance of an intimate and detailed knowledge of the medical beliefs of their patients. Younger men are inclined to laugh and scoff at the silly notions so many of their patients have. But they soon learn that without a thorough knowledge of these beliefs, medical practice becomes a cold body from which the spirit had departed; and that any attempt at ridicule will soon estrange their clients.

In social medicine a knowledge of these beliefs is even more important because it impinges on the province of health education, a function of health centres which differentiates their work from that of ordinary outpatient practice. It is necessary to wean the people away from their false beliefs and away from the old people who keep these beliefs going.

We have therefore had to make a study of these beliefs not merely for academic reasons but also because our work can start only when reason has replaced the black mist of superstition and utter credulity. For this reason we have put up a sheet of paper in every service-room of the Health Centre to enable the health workers to record the beliefs as they stumble across them. This method of recording has functioned very well but it has also had an amusing sequel. One woman had been assiduously practicing a particularly obnoxious belief until we found out that she had seen it on the wall chart and had been trying it out in good faith!

One could write a volume on these false beliefs and their variations. For the sake of conciseness I must of necessity confine myself to the principal ones and to comment on them. They resolve themselves under the following headings:

1. *Food and Disease:* (*a*) *Too much meat and/or bread causes worms.* This belief is very prevalent and pernicious as children are often denied their share of the meat. Bread is given in any case, otherwise there would be nothing else to give. It takes many talks to make mothers understand that the children pick up the worms in the backyards where the lavatory bucket contents have to be buried through lack of a systematic collecting system by the local authority. In such cases we very often withhold worm treatment until the householder had decided to dig a compost-pit and thus had made a start to decontaminate the soil in his backyard.

(*b*) *Squashes contain valuable foodstuffs.* In their misguided attempts to comply with the slogan 'eat more vegetables' the people fasten on to squashes and this is given to babies of a few months old resulting in severe insult to their colons.

(*c*) *Lemons dry up the blood.* Mild forms of malnutrition are very common and it is a pity that such false beliefs should keep such a good source of vitamin C away from the people.

(*d*) *Guavas are unhealthy as they cause appendicitis.* They are afraid that the pips may lodge in the appendix and so another valuable fruit goes by the board.

(*e*) *Maasbankers are not fit for decent people to eat.* These fish are cheap, rich in vitamin A containing fats and delicately flavoured when cooked fresh. But because white people do not want to eat maasbankers there must be some stigma attached to them.

(*f*) Certain patent baby foods are used not as foods but because of a mistaken idea that they possess valuable medicinal qualities. These expensive and often unnecessary foods make drastic inroads on the meagre incomes of these people.

(*g*) *In winter-time because of the dew, tomatoes are dangerous to give to children.* Similar beliefs concern many other vegetables and thus valuable protective foodstuffs are withheld.

*Teeth:* (*a*) Ash is often used as a dentrifice. This is apt to cause a *gingivitis.*

(*b*) *If you pull teeth out during pregnancy, the baby will be born crippled.* Malay mothers are apt to believe this fable and it is very difficut to convince them of the benefits of dental supervision.

(*c*) *Teeth should not be extracted in winter as you may get a cold in them.* This belief is connected with the general 'koue' theory—'koue op die niere', 'koue op die maag' and so on. People thus deny themselves the benefits of early dental treatment.

(*d*) There is an overwhelming demand for *extractions.* Fillings are frowned upon. Young people often ask for total clearances for no reason at all.

(*e*) 'Stillbirths and abortions may result from teeth extractions during pregnancy'.

(*f*) Cavities are often filled with silver paper, match-sticks, leaves, cotton wool, etc. The result is often a *tooth abscess.*

(*g*) 'Extraction of front teeth will cause fits and madness'.

(*h*) One should not eat before going to the dentist'. Faint-

ing fits are common. This belief probably derives from the injunction not to eat before an anaesthetic.

*Notions about Babies:* (*a*) *Castor oil should be given to all babies and children weekly to clear their stomachs of dirt.* This is often the cause of an unexplained diarrhoea. Apart from reasoning with the mother, I often tell them that if castor oil were necessary, the Father of us all would have sent a bottle of oil along with the baby.

(*b*) *When the periods come back, the mother's milk is bad for the baby.* On this account babies are often weaned at a very tender age.

(*c*) *Babies heads should not be washed while the bones (fontanelles) are still soft.* The condition of same babies' heads can therefore be left to the imagination. Sores and impetigo are common.

(*d*) Amulets are very often seen in the nature of 'electric collars', coins, little bags of garlic or spice, etc. They are worn to keep away disease and to *keep off the Evil One.*

(*e*) Many mothers firmly believe that babies are born with worms and that therefore it is 'natural' and nothing can be done about it.

(*f*) Mothers are fond of swaddling their babies in tight abdominal binders for as long as 3 to 6 months after birth. It contributes to *weak abdominal musculature.*

(*g*) Mothers' milk is often squirted into babies' eyes when they become infected.

(*h*) A sweat rash in a baby is usually ascribed to *acid in the blood.*

(*i*) *Die kent se tongetjie sit vas* is a very common belief and mothers are often very much offended when we tell them that it is a very rare occurence, indeed.

*Births:* (*a*) *The after-birth will come away more easily if you blow on a bottle.* There is probably an element of truth in this.

(*b*) *If you shave the mothers' pubic hair the baby will be born without eyebrows or lashes.*

(*c*) *The cord will not separate if the antenatal mother sews or combs her hair in bed.*

(*d*) Gamps love lubricating the passages with oil or grease to facilitate the passage of the child. Fortunately puerperal sepsis is not often seen.

(*e*) *Bowel washouts must not be given before labour starts as this will cause a stillbirth.*

*Doctors, Hospitals, etc.:* (*a*) The people have a great fear of hospitals in general. Terrifying tales are told by patients on their return from hospitals. The general attitude is: 'Nee, ek gaan liewer by die huis dood!'

(*b*) Children are instilled with a fear of doctors and dentists.

(*c*) They are peculiarly afraid of injections whereas Natives, as is well-known, do not mind injections and even demand them for even the most trivial of complaints.

(*d*) *Injections must not be given in the arm as it is too near the heart.*

(*e*) *Castor oil applied to the head is good for headaches and colds.*

(*f*) *Red flanelette is good for all chest conditions.*

(*g*) Oily medicines are demanded for chest conditions *to oil the lungs and to soften the cough'.* This is a good opportunity to give vitamin oil!

(*h*) Some people have the curious idea that blood taken for the Wassermann reaction is used for blood transfusion purposes.

(*i*) The belief that intercourse with a virgin will cure a dose of gonorrhoea is not very common.

(*j*) The belief that one should only consult a doctor or dentist when one is sick or has a toothache, is all too prevalent. Many older women are not eager to take a young midwife for their confinements.

*Remedies:* (*a*) Paraffin oil, green 'alsblare', raisins, cobwebs, dogs' hair, grease and oil, etc., are used for cuts, sores and burns. Some of these 'huisraadjies' are harmless but some are injurious, e.g., paraffin is commonly applied to cuts resulting in an acute dermatitis. This detrimental result is then felicitously ascribed to the 'gif wat trek'.

(*b*) In case of fever the whole body is packed with 'alsblare' steeped in motor-car oil. In acute chest conditions a cat is hastily slaughtered and the warm skin is applied to the chest. When tail-fat is used and it turns brown, it shows that the fever has been driven out. They love to apply things 'om die gif uit te trek'.

(*c*) *For sunburn rub on the juice of sourfig leaves.* For all I know this may be a useful remedy!

(*d*) *Goat manure given by mouth is excellent in measles.*

(*e*) *To stop hiccups, put match-sticks in the ears.*

(*f*) *Convulsions in children are best treated by applying mashed pumpkin to the limbs.*

(*g*) *Diphtheria can be cured with borax and honey.*

(*h*) Cloves and spices in a bag are often applied to the abdomen of children suffering from gastro-enteritis. They will try all sorts of remedies for days on end and when we see the child it is usually *in extremis.*

(*i*) *In severe stomach pain give wonderkroon essence and apply a tight binder.*

Many of these remedies are harmless. Some however are not only harmful but also make for complacency with consequent delay in seeking medical aid. When asked why they use these absurd remedies, the reply is usually 'hulle het so gesê'. Old women and grandmothers take a special delight in 'doctoring' and they are therefore very influential in the community. Many of the younger people have had so little schooling that they are eagerly fastened on to by these elderly houris.

*Miscellaneous Beliefs:* (*a*) Soap is very often used for 'B.O.', i.e. for underarm perspiration and a rash is a common result.

(*b*) Headwashing is not favoured in times of illness, the menses and for nearly any excuse. The result is impacted dirt and vermin with secondary infection, sores, impetiginization, scratching, adenitis, stiff necks and a general toxaemia which slowly saps the strength and stamina of the child.

(*c*) Newspaper advertisements are treated as gospel truths; happily newspapers these days exercise great discretion in placing such medical advertisements.

(*d*) *Sterility is the fault of the woman, never of the man.*

(*e*) *Coughing may be stopped by raising the arms above the head.*

(*f*) Any kind of glasses is better than none at all. Spectacles are often handed down from mother to daughter. Generally speaking the coloured girls are not so keen on glasses as the Native women.

(*g*) The principal meal is taken at night when the bread-winner comes home. It means that during the day the children subsist mostly on coffee and bread and jam. The school-meal is a real godsend.

(*h*) *Nose-bleeding may be stopped by tying the thumbs with string.*

(*i*) *Green leaves pushed into the ears will stop toothache.* Unfortunately the usual result is an external otitis.

(*j*) *Conception can be prevented by drinking a glass of cold water immediately after sexual intercourse.*

(*k*) Tuberculotics invariably use a pan with sand to spit in. The flies love it!

(*l*) *Failing eyesight is cured by piercing the ear lobes.*

(*m*) *Worm treatment should only be given at the decline of the moon.*

(*n*) *Dutch medicines are good because the label on the bottle says so.*

(*o*) *Cancer can be cured by having lots of babies.*

*General Conclusions:* These false medical beliefs are not only interesting but a sound knowledge of them is essential in dealing with these people. Invariably causation of disease, diagnosis and treatment are in no small measure affected by these beliefs. They also enter into almost every other aspect of their daily lives. They lead them into incurring unnecessary expenditures; serious cases are withheld from prompt medical attention; they sometimes lead to break-up of family life; they contribute to the already high infantile mortality; they induce hysterical states and other forms of neurosis.

These beliefs are an expression of the abysmal ignorance of the people, of their misery and unhappiness. The cure is health education to which every general practitioner should contribute.

I wish to thank the Secretary for Health for his permission to publish these notes.

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